PHILADELPHIA NEUROLOGICAL SOCIETY.

March 22d, '97. President, Dr. Charles W. Burr, in the chair.

Dr. A. A. Eshner exhibited a patient with BASILAR MENINGITIS. (See this journal for March, p. 167.)

D. Chas. K. Mills.—The case, I think, is rather difficult for diagnosis. I saw this man a year or so ago. From the symptoms then presented the diagnosis of a form of chronic degeneration of the nervous system seemed to be justified. I reported the case as probably one of posterior sclerosis of aberrant type. The man has no headache. He has loss of hearing, which is gradually progressing, and which we sometimes see in cases of chronic degeneration of the nervous system. It is not impossible that this is one of those cases of chronic degenerative disease which will finally end as a case of paralysis of the insane.

Dr. Charles K. Mills reported five cases of

HEREDITARY TREMOR,

and exhibited one of the patients.

Case 1. Mrs. E. P., forty years of age, presented herself at the neurological service of the Woman's Medical College. The patient has had tremor in both upper limbs, greater in the left, since her earliest childhood. The tremor is increased by excitement or by grasping an object, though when she is in repose there is scarcely any tremor in her hands. The tremor in the head has only been noticed for eight or nine years, and is attributed to a fright from a man supposed to be insane. This tremor in the head is greater than in the hands, and is of a slightly rotary character. The patient has never noticed any tremor in the lower limbs. She is very excitable. The knee-jerks are present but not notably exaggerated. There is no ankle-clonus. The sensation for touch and pain is normal in the entire body. There is no inguinal tenderness. Inframammary tenderness is found on both sides; is a little more intense on the left, but the whole area of the stomach is tender to pressure, and the patient suffers from indigestion. There is no tenderness along the spine, and no limitation of the visual fields. No history of the parents, and no statements in regard to tremor in the father's family, can be obtained. The mother's family is not known.

The daughter has had a similar but more intense tremor. noticeable only in her hands, ever since birth. Her head is not affected. She is not as excitable as her mother. She has a tremor at all times, even when not excited. She has a child of four years, who has no tremor.

Case 2. T. J. E., forty-five years of age, a resident of Mechanicsburg, Pa., was seen with Dr. J. H. Boyer.

Since about 1882 he has had a peculiar jerking and tremulous movement of the head, which has remained almost unaltered. This tremor has a tendency to draw the head backward and to the right. The chin and whole head turn to the right. Some tremor of both hands and arms is also observed. Any occupation increases the tremor of the head. He occasionally takes a drink of whiskey to steady himself. He has no nystagmus, and the special senses apparently are not affected; the knee-jerk is a little plus.

The father of the patient, now eighty-five years of age, has a similar tremor of the head, which developed when he was sixty-five. A sister, of forty-two years, is similarly affected, and in her case the tremor was first noticed at the age of thirty-two or thirty-three.

Dr. A. A. Eshner exhibited a case of

CONGENITAL TREMOR.

He stated that persons occasionally present persistent tremor without obvious cause or other morbid signs. Sometimes the shaking may be observed in several members of the same family, and there may be an evident transmission from parent to children. The tremulousness may not appear until late in life or it may develop early. The earlier the manifestation, the stronger would be the reason for suspecting a structural or organic basis. On the other hand, slight tremor in an infant might readily be obscured by the natural incoordination of movement present at this time of life. Apart from the rotary and nodding movements of the head, not rarely seen in rachitic children, tremor is not often observed in infants in the absence of structural or organic disease.

The case which he reported was designated as one of congenital tremor on account of the early period in life at which the movements were noted—with the possibility of its still earlier presence; of the absence of evidence of an organic cause, and of the freedom of other members of the family from a similar disorder.

M. F. M., now thirty-six years old, presented himself in the autumn of 1893, at the Infirmary for Nervous Diseases in the clinical service of Dr. S. Weir Mitchell, complaining of a tremulousness that had been present as long as he could remember. His recollection carried him back to boyhood, when the right hand shook distinctly in school work. He was unable to state with certainty whether or not both hands were tremulous, but he thought they were. No tremor had been noticed by his parents during infancy. For perhaps fifteen years he had noticed a certain weakness of the legs, which would at times give way in walking, and staggering result. This was particularly marked in ascending acclivities. Such muscular efforts were unattended with tremor, although for a period of eight or ten years, when one leg was thrown over the other, a shaking—possibly clonus, but more probably simple tremor—would be set up in the foot.

The symptoms noted gradually progressed in intensity. For a moderate period of the time the patient, in the pursuit of his occupation as a laborer in a mill, had been obliged occasionally to carry heavy loads, and it was noticed that the tremor was increased after such efforts. The tremor was greatly aggravated by observation, by unusual surroundings, by emotions, by excitement, by laborious work, and by the chewing and smoking of tobacco. It was absent during sleep, and as a rule during rest, and was essentially an intention tremor. The drinking of beer would for a short time be followed by a lessening of the movement, with, however, subsequent aggravation. The trembling was worse when constipation existed, and also when diarrhea was present. The patient related that, as a child eight or nine years of age, he would from time to time be frightened by the thought of a strange person in his bedroom, and he would not be quieted except by the presence of his father, although he slept with an older brother. In disposition he was irritable and easily angered.

In standing, the patient appeared quite steady, although he felt a slight shaking of the right hand. The movement was quite beyond his control, and was increased by efforts to suppress it. When the arms were extended at right angles with the trunk, the fingers and hands were set into rhythmic tremor, as if from alternate action of the flexors and extensors of the carpus. Muscular activity was soon followed by fatigue, while support afforded subjective relief. On attempting to touch the tip of the nose or of the ear with the fingers, and on attempting to bring the fingers of opposite hands together, coarse oscillatory movements took place, rendering variable and uncertain the result, which was sometimes successful, sometimes not. The knee-jerks were active, full and strong, rather exaggerated than otherwise. Dorsal flexion of the foot on the leg elicited only an abortive attempt at ankle-clonus. There was no derangement of sensibility, common, painful or thermal. The pupils were equal, regular, and reactive to light. Nystagmus was not present. Speech was not interfered with. There was no evidence of disease of heart or lungs. The appetite was good. The tongue was coated slightly, and protruded in the median line; it shared in the general tremor, particularly at its tip. Costiveness was usually attended with nausea, acidity and vomiting. Hearing was impaired upon the right side. Memory for recent events was enfeebled, that for remote events preserved.

The patient had suffered from enteric fever at the age of twenty-five years. He denied sexual excesses, and had no knowledge of venereal infection. During a period of twelve years, until within a short time of coming under observation, the patient had indulged periodically in alcoholic excess. He had used tobacco freely, but had taken tea and coffee in moderation. There was no history of blow, nor injury, nor traumatism of any kind. The father of the patient had been killed in a railroad accident. The mother was living at the age of sixty-five or sixty-six years; she was believed to be rheumatic, and suffered from time to time from attacks of precordial pain, which were relieved by the ingestion of a glass of water. One brother was living and well; two brothers were dead of enteric fever—one of these suffered from stuttering speech.

The patient's handwriting showed the pronounced tremulousness, while that of the brother showed nothing abnormal. Tracings of the patient's tremor were noteworthy for their want of uniformity and of regularity, although the rate of movement appeared to have been about six per second. The movement in the two hands was approximately, if not absolutely, synchronous.

It is not impossible that there exists in this case a degenerative or sclerotic process, necessarily slight in degree, involving the lateral columns of the spinal cord, although the symptoms are scarcely well enough defined to render such a conclusion at all certain. As opposed to this diagnosis must be considered the absence of ankle-clonus and of other spastic phenomena, and of noteworthy muscular disability, as well as the early onset and the slow progress of the symptoms. Multiple sclerosis may be excluded from the absence of nystagmus and of peculiarity of speech.

Dr. Chas. K. Mills exhibited a case of

CEREBRAL DIPLEGIA.

J. M. C., a white girl of eighteen years, was born with instruments after a very hard labor, and for some time succeeding birth was supposed to be dead. She first walked when she was fifteen months old, and it was noticed that she dragged the right foot, and that she waddled when she ran. When she was eight years old she had "scarlet fever and measles at the same time," which was followed by a partial loss of voice. A slight trembling of the limbs was noticed before the attack of scarlet fever, in fact, the mother of the patient cannot state just when the tremor was first observed. There is no history of a similar condition in any branch of the family, and the other children are well. The patient has had no convulsions, and has made fair progress in school.

Present condition—She is a well-nourished, intelligent young woman. The tremor is most marked in the head and right arm. The movements are coarse and jerky in character; those of the head being lateral, anteroposterior and half-rotary. The head, trunk and extended arm seem to move together. The tremor of the right upper extremity involves the whole arm, and is partly lateral and partly rotary, and is most marked in the hand. The left upper extremity is affected with tremor of the same character, but less in degree. The left lower extremity is more affected than the right with the same coarse tremor, lateral and rotary in direction, and more marked distally. The knee-jerks are plus on both sides. Ankle-clonus is found on both sides, and is especially marked on the right, where it is of the persisting variety. The right foot is dragged slightly when the patient walks. The hands are cold and dusky; the feet also are usually cold. The right leg is about one-half inch shorter than the left, and smaller throughout.

Dr. A. A. Eshner.—I have had the opportunity of seeing the girl shown by Dr. Mills, at the clinic of Dr. S. Weir Mitchell. The case was looked upon as one of cerebral birth traumatism. To exclude the possibility of chorea, the girl was treated for a month by the hypodermic use of Fowler's solution in ascending doses, and under this treatment she did not make the slightest improvement.

Dr. Wm. G. Spiller.—The history of difficult labor, the dragging of one foot in walking, the retardation of growth, the bilateral ankle-clonus, etc., are in favor of the diagnosis of diplegia with greater involvement of one side. The tremor has nothing of the character of chorea, and we may probably dismiss all thought of this disease. It seems to have become much more evident after the attack of scarlet fever, when the child was eight years old. After acute affections it is not uncommon to see the development of organic nervous disease. It is, therefore, not surprising that tremor should develop in this case after measles or scarlet fever. I am inclined to think that the tremor is due to a lesion at birth, especially as I have studied a case in which a cortical sclerosis was produced probably in the first years of extrauterine life and was followed by the development of epileptic convulsions at the age of ten. A similar case of infantile paralysis with development of epileptic attacks about this same age, has been reported by v. Monakow. I am inclined to think that the tremor in Dr. Mills' patient is a part of the diplegia, and that the infectious disease at the age of eight was only the indirect cause of its development; in other words, that it was an "*agent provocateur*."

Dr. A. G. Thomson read a paper on

ISOLATED, TRAUMATIC, BILATERAL PARALYSIS OF THE ABDUCENS NERVE.

A man of forty-three years, with no history of specific or spinal disease, a year and a half ago fell a distance of thirty-three feet, from a scaffolding to the ground. He regained consciousness after an hour and a half, and found that he had complete bilateral paralysis of the sixth nerve, and that the right side of the body was bruised. There were no signs of fracture of the skull, nor of injury of any other cranial nerves. He recovered from his bodily injuries within a few days, and was able to return to his work. The bilateral ocular paralysis is still complete. Dr. Thomson thought the lesion was caused either by a bruise or tear of the nerves in their course from the pons to the sphenoidal fissure.

Dr. F. S. Pearce.—I have seen reported a case of bilateral paresis which the author claimed was probably due to a dystrophy of the external recti muscles. The condition was congenital. Dr. Thomson's explanation is quite plausible.

Dr. Wm. G. Spiller.—Cases of sudden paralysis of the sixth nerve from unknown causes have been reported, but trauma seems to have produced the paralysis in the patient described by Dr. Thomson. I agree with him that the lesion was probably not nuclear. It is possible that a small hemorrhage might destroy both sixth nuclei, but it is difficult to understand how the seventh nerves could entirely escape in such a lesion. The knee of the seventh nerve embraces the nucleus of the abducens very closely, and for a long time was thought to receive fibres from it. I am more disposed to think the lesion is probably at the point of exit of the sixth nerves from the bulbopontine junction, at which place they are close together.

Dr. Joseph Leidy read a paper entitled

NEURALGIA OF THE CRANIAL NERVES AS A SYMPTOM OF MALARIA.

He stated that occipital neuralgia has frequently been observed as a symptom of uremia, especially the uremia resulting from the atrophic form of chronic diffuse nephritis.

The first case was one of arteriocapillary fibrosis. During the first year there were gastrointestinal disturbances; during the second year, neuralgia, principally of the occipital, inferior dental and sciatic nerves, and evidences of renal involvement, albumin and low specific gravity of the urine; during the third year involvement of the cranial nerves with evidences of cerebral affection, *i. e.*, paresis rather than paralysis, of the tongue and vocal cords, and periodic attacks of dysphagia and dysphasia.

Finally the cardiac action became insufficient. The respiration during eight days was of Cheyne-Stokes character, but the patient rallied and was able to go about without assistance, and to attend to business. The Cheyne-Stokes respiration was noticed at intervals afterward, and the patient finally succumbed to his disease.

Certain of the symptoms were attributed to the contracted kidneys; others to interference with the cerebral circulation at the base of the brain. The speaker considered the temporary recovery after the attack of Cheyne-Stokes respiration of interest, and referred to a similar case in which the recovery was permanent. He spoke especially of the slowly developing nephritis as a part of a general arterial degeneration without changes, for a time, in the renal excretion, and of the dysphasia due to paresis and incoordination of the movements of the tongue.

The second case well illustrates the importance of microscopic examination of the blood in cases of sudden loss of consciousness.

The patient was admitted to the hospital in an unconscious condition. An examination showed albumin and high specific gravity of the urine. The suspicion of uremia was aroused. The microscopic examination of the blood revealed the plasmodium malariæ in great abundance. There were ameboid and pigmented forms and several flagellated bodies. The patient was soon seized with severe rigors. In addition to treatment directed to the kidneys, quinine in ten-grain doses was given every hour until a drachm had been administered. Improvement was observed within twenty-four hours; consciousness returned, and the patient made an uninterrupted recovery.

The history was as follows: The man had left the swamps of North Carolina to place himself under treatment in Philadelphia for obstinate neuralgia and vertigo. He grew rapidly worse while on the vessel, and after arriving in this city fell unconscious in the street while on his way to the hospital. The speaker regarded the case as one of malarial nephritis.

Dr. James Tyson.—The frequent association of neuralgia with Bright's disease, especially neuralgia of the posterior auricular nerves, is a recognized condition; at the same time the independent occurrence of neuralgia due to other causes is so frequent that I should hardly consider this symptom of diagnostic value.

The first case, it seems to me, is admirably explained by supposing that there was a primary general arteriosclerosis in which the vessels of the kidneys shared ultimately with the other blood vessels, and that this sclerosis was responsible for the lesion in the internal capsule and also for the renal symptoms which came on as a later manifestation of the disease. It is often a nice question for diagnosis whether the renal disease is primary and the vascular disease secondary, or whether the vascular disease is primary, involving more or less the blood vessels of the system and the kidneys. I think that this latter condition is found to produce other symptoms prior to the renal symptoms, as was the case in Dr. Leidy's patient.

CONCERNING THE HISTOLOGICAL CONDITION OF THE PERIPHERAL NERVES AND THEIR CENTRES AFTER SECTION. Wiener klinische Wochenschrift, April 29th, 1897. By Arthur Biedl.

With the exception of the first paper published by Nissl in 1890, we have no detailed statements in regard to the changes which occur in the cell body after section of the nerve fibre (Biedl). Biedl has operated on three dogs and three rabbits by removing a portion of the sciatic nerve in the upper part of the thigh. The animals were killed at different periods after the operation, and their spinal cords and sciatic nerves were examined. The results of his investigations are as follows:

Degenerative changes may be found three days after section of the nerve in the cells from which these fibres arise. These changes consist in rarefaction and coarse, granular disintegration of the chromatic substance. At the same time degenerative changes may be observed in the peripheral end of the nerve, while the central end except at the point of section, remains normal. Two days later the granular disintegration of the cell body is more distinct, and fine granules may be seen. The first traces of degeneration may be noticed in the intramedullary portion of the anterior roots. Eight or nine days after the section the ganglion cells present the appearance of the fine, granular, so-called molecular disintegration. The destruction of the cell obtains its greatest degree about the eighteenth day, and consists of fragmentation of the cell body and of transformation of the fine, granular into homogeneous degeneration. The degeneration of the peripheral end of the cut nerve at this time is no longer active, but nearly the whole central end presents the appearances of beginning degeneration. After twenty-eight days the cells are more deeply stained and are homogeneous, but with indications of the formation of cellular processes and nuclei. These are probably the first manifestations of the process of regeneration. SPILLER.